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Review

When insomnia is not just insomnia: The deeper correlates of disturbed sleep with reference to DSM-5

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ABSTRACT

Recent scientific evidences have brought a paradigm shift in our approach towards the concepts of insomnia and its management. The differentiation between primary and secondary insomnia was proved more hypothetical than actual and based upon the current evidences insomnia subtypes described in earlier system have been lumped into one-insomnia disorder. Research in this field suggests that insomnia occurring during psychiatric or medical disorders has a bidirectional and interactive relationship with and coexisting medical and psychiatric illnesses. The new approach looks to coexist psychiatric or medical disorders as comorbid conditions and hence specifies two coexisting conditions. Therefore, the management and treatment plans should address both the conditions.

A number of sleep disorders may present with insomnia like symptoms and these disorders should be treated efficiently in order to alleviate insomnia symptoms. In such cases, a thorough history from the patient and his/her bed-partner is warranted. Moreover, some patients may need polysomnography or other diagnostic tests like actigraphy to confirm the diagnosis of the underlying sleep disorder.

DSM-5 classification system of sleep–wake disorders has several advantages, e.g., it has seen insomnia across different dimensions to make it clinically more useful; it focuses on the assessment of severity and guides the mental health professional when to refer a patient of insomnia to a sleep specialist; lastly, it may encourage the psychiatrists to opt for sleep medicine as a career.

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Recently, in the Diagnostic and Statistical Manual-5th edition (DSM-5), the definition of insomnia disorder has been changed (American Psychiatric Association, 2013). The major shift has been the omission of the terms "primary" and "secondary" insomnia and these have been lumped under the diagnosis of insomnia disorder (Reynolds and O'Hara, 2013). This is due to the fact that insomnia has been recognized as an independent disorder that requires precise definition and specific treatment, even when it is comorbid with other psychiatric and medical conditions (Roth, 2009). There are a number of sleep disorders which may present with complaints similar to that of insomnia and if we do not recognize them, it may hinder effective treatment.

This paper describes the recent changes in definition of insomnia with reference to DSM-5 and reviews the sleep conditions that can overlap with insomnia. We will also discuss the relevance of changes for the clinical practice.

1. Insomnia diagnosis in present classification systems: similarities and differences

Earlier classifications e.g., Diagnostic and statistical Manual-IV Text revision (DSM-IV-TR), International classification of diseases-10th (ICD-10) and International Classification of Sleep Disorder-2nd edition (ICSD-2) have categorized the insomnia into primary and secondary forms (American Psychiatric Association, 2004; World Health Organization, 1992; American Academy of Sleep Medicine, 2005). For example, the DSM-IV-TR described 'primary insomnia' as a disorder of either initiating or maintaining the sleep, or non-restorative sleep for more than 1 month (American Psychiatric Association, 2004). In addition, the sleep disturbance should have imparted significant impairment in daily functioning and most importantly, it should not have occurred during the course of medical or psychiatric disorders, including periods of substance abuse or withdrawal (American Psychiatric Association, 2004). Insomnia associated with latter conditions was considered "secondary insomnia".

The World Health Organization, in the ICD-10 classification, added "early morning awakenings" to the definition of insomnia as per DSM-IV-TR and described quantitative specifiers which mandated that the sleep problems must occur on 3 or more nights a week (World Health Organization, 1992; American Psychiatric Association, 2004). In addition, it indicated that, along with the impairments that were already described in DSM-IV-TR, individuals with insomnia should have excessive concerns about the symptoms. Instead of 'secondary insomnia' it described a category of "organic insomnia" which could be diagnosed when insomnia occurred in the presence of other medical, neurological or psychiatric disorders (World Health Organization, 1992).

The dichotomy of 'primary' and 'secondary' insomnia was also preserved in the ICSD-2 having been proposed by American Academy of Sleep Medicine in 2005. Unlike DSM-IV-TR and ICD-10 however, it described four types of primary insomnias: idiopathic, adjustment, paradoxical and psycho-physiological (American Academy of Sleep Medicine, 2005). It also described multiple types of secondary insomnias that were due to other conditions. These conditions included substance abuse or withdrawal, medical or psychiatric disorders, environmental conditions and poor sleep hygiene (American Academy of Sleep Medicine, 2005). Thus, we can see that the definition of insomnia differed across all the major classification systems.

As a consequence of the epidemiological, clinical and neurobiological evidences which have evolved in recent years, a paradigm shift has been introduced in the definition of insomnia by the American Psychiatric Association in its recent classification system – DSM-5 (American Psychiatric Association, 2013). The major changes include the removal of the dichotomy between primary and secondary insomnia and an addition of quantitative specifier (American Psychiatric Association, 2013). The new classification system aimed to move away from the causal relationship integrated in the DSM-IV-TR and to introduce a bidirectional and interactive relationship between insomnia disorder and coexisting medical and psychiatric illnesses. Specifically, instead of separating insomnia subtypes, the DSM-5 defines only "insomnia disorder" which can be "primary" or "co-morbid" with other psychiatric or medical conditions (American Psychiatric Association, 2013). In this system, insomnia is viewed as a comorbid disorder that warrants separate treatment attention (American Psychiatric Association, 2013). Furthermore, it is now recognized that a number of sleep disorders can mimic insomnia, and, to provide more accurate diagnoses and better care to the patient, the definition of insomnia must rule out the presence of these conditions (American Psychiatric Association, 2013). In the following sections, we will discuss both of these issues in detail and will try to examine the scientific evidences and rationale of these changes.

2. Primary versus secondary insomnias

In the traditional view, insomnia was considered a symptom of psychiatric illnesses (Sánchez-Ortuño and Edinger, 2012). According to this view, it was expected that insomnia should improve with the treatment of psychiatric disorders. However, the usual treatments for psychiatric disorders do not typically address the insomnia, with the consequence that for many patients their insomnia would persist even after their psychiatric symptoms had abated (Sánchez-Ortuño and Edinger, 2012; van Mill et al., 2010). The reason for the resistance of insomnia to non-sleep interventions is that insomnia is maintained by sleep-related, non-adaptive cognitions and behaviors that are sleep specific and are independent of the comorbid condition (Taylor et al., 2005; Buysse et al., 2008; van Mill et al., 2010). Cognitive behavioral therapy for insomnia (CBT-I) specifically targets these insomnia perpetuating cognitions and behaviors and thus effectively alleviates insomnia in the presence of comorbid psychiatric and medical conditions (Watanabe et al., 2011; Shimodera et al., 2011; Sánchez-Ortuño and Edinger, 2012; Wagley et al., 2013).

3. "Primary insomnia" subtypes: is differentiation actual?

As already mentioned, four different types of primary insomnias have been described in ICSD-2, idiopathic, adjustment, paradoxical and psycho-physiological (American Academy of Sleep

Medicine, 2005). However, research suggests that the differentiation between them is more artificial than actual (American Psychiatric Association, 2013; American Academy of Sleep Medicine, 2014). Resultantly, the sub-classification of primary insomnias has not been included in DSM-5 and removed from the International Classification of Sleep Disorders-3rd edition (ICSD-3) (American Academy of Sleep Medicine, 2014).

ICSD-2 defined adjustment insomnia as a disorder that appears during the stressful situations and remits as soon as the exposure to stress is terminated. However, the diagnosis needs to be changed if the symptoms persist for more than 3 months. Similarly, psycho-physiological insomnia was diagnosed when the sufferer had a learned behavior and cognitive distortions that was maintaining the insomnia; in the state of paradoxical insomnia, the sufferer had mis-perception regarding his sleep and it was diagnosed when the sufferer had under-estimation of sleep duration or depth; lastly, idiopathic insomnia was considered a chronic idiopathic condition. Among all of these four, DSM-5 has retained the diagnosis of adjustment insomnia but it has described it as situational insomnia (American Psychiatric Association, 2013).Certain people are more predisposed to the situational change and they develop insomnia during these periods (Bonnet and Arand, 2010). However, with time, they often adopt a maladaptive behavior, and may fulfill criteria for other primary as well as secondary insomnias e.g., psychophysiological, sleep-state misperception, poor hygiene and comorbid psychiatric disorder (Fernandez-Mendoza et al., 2011; Gupta and Lahan, 2011). The electrophysiological studies have also shown that hyperarousal is seen in both paradoxical insomnia and psycho-physiological insomnia (Turcotte et al., 2011; Bastien et al., 2013, 2008). Thus, the difference between different primary insomnias is more superficial than actual. This is one of the reasons that categorization of insomnia into different sub-types has been questioned by DSM-5 (DSM-5).

Subjects with chronic insomnia, irrespective of the subtype, often misperceive their sleep and have a subjectively less sleep as compared to objective duration (Fernandez-Mendoza et al., 2011). This results in anxiety and poor sleep hygiene that perpetuates the insomnia (Unbehaun et al., 2010). In other words, in these patients, mental-fatigue is not sufficient to inhibit the hyperarousal and despite high cognitive load during night they are not able to catch the sleep (Pérusse et al., 2013). To deal with this issue, (CBT-I) includes the elements that address all types of factors – stimulus control therapy, relaxation, sleep restriction therapy and improvement of the sleep hygiene which are used in various combinations, depending upon the requirements (Unbehaun et al., 2010).

4. Sleep disorders presenting with complaints similar to insomnia

In addition to being a comorbid condition with a number of psychiatric and medical disorders, insomnia is also frequently associated with other sleep disorders (Becker and Novak, 2014; Al-Jawder and BaHammam, 2012). Furthermore, there are sleep disorders that present with the symptoms of insomnia. This may result in a situation, in which treatment of insomnia is provided to the patient, when, in fact, there is a chronic, underlying sleep disorder that remains undiagnosed and untreated. This problem has been recognized in the DSM-5 and the ICSD-3 which have specified the conditions that need to be differentiated from insomnia (American Psychiatric Association, 2013; American Academy of Sleep Medicine, 2014). In order to differentiate between insomnia and these other sleep conditions, clinicians need to understand the specific and common features of insomnia and other sleep disorders. These disorders include sleep disordered breathing, restless legs syndrome, narcolepsy and circadian rhythm disorders and parasomnias (DSM-5).

5. Sleep disorders that present with complaints of insomnia

5.1. Insomnia and sleep disordered breathing (SDB)

Sleep disordered breathing is characterized by recurrent episodes of cessation of breathing or hypoventilation (American Academy of Sleep Medicine, 2014). This group of conditions include, for example, obstructive sleep apnea and central sleep apnea. These particular types of disordered breathing occur so frequently and have such a broad range of effects that they are considered separately below.

5.2. Insomnia in obstructive sleep apnea (OSA)

Obstructive sleep apnea is defined as cessation of breathing for at least 10 s during sleep in the presence of continuous respiratory efforts (Fig. 1A). Affected individuals usually remain asleep, but the sleep is fragmented and characterized by numerous awakenings throughout the night. A related phenomenon is hypopnea during which breathing is reduced but is not completely interrupted. Concurrently there is decrease in the oxygen saturation of the blood. For clinical purposes, the rate of apneas and hypopneas indicate the severity of the breathing disorder.

There is a frequent (22–54.9%) comorbidity between OSA and insomnia (Al-Jawder and BaHammam, 2012; Alotair and Bahammam, 2008).The recurrent apnea/hypopnea events and post-event arousals can produce sleep fragmentation and frequent awakenings throughout the night (Fig. 1A). These occurrences in turn may worsen the quality of sleep and result in sleep maintenance insomnia (Al-Jawder and BaHammam, 2012; Alotair and Bahammam, 2008). Nocturia i.e., waking at night one or more times for voiding is another factor that can disturb sleep in OSA patients. Thus, some OSA patients may present with insomnia, but the symptoms have been caused indirectly by a more primary condition.

Gender is another factor that influences the frequency and type of insomnia symptoms that occur among OSA patients; specifically insomnia is reported more frequently by female patients with OSA as compared to males (Alotair and Bahammam, 2008; Lavie, 2007). Among women with OSA, sleep-onset insomnia is the predominant type of insomnia, as opposed to the predominantly sleep maintenance issues described in males (Alotair and Bahammam, 2008; Lavie, 2007). Early-morning awakening insomnia can also be present, and is associated with daytime sleepiness (Chung, 2005; Wickwire et al., 2010).

While OSA patients frequently complain of insomnia, there is also a high prevalence of OSA reported among patients with chronic insomnia. Among elderly patients with insomnia the rate of OSA is alarmingly high (43–67%) (Lichstein et al., 1999; Guilleminault et al., 2002). These findings thus support the recommendation that older adults with insomnia should have a sleep study for the purpose of detecting the presence of an underlying OSA condition. A number of tools are now available for screening of obstructive sleep apnea and they may be used in clinical practice. These are shown in Table 1 below.

When insomnia appears in the context of OSA, the physician is confronted with a choice of several treatment options. One option is that the OSA is treated first and if the insomnia persists, a targeted insomnia treatment follows. Alternatively, the insomnia can be treated first, or in conjunction with the OSA treatment. This latter method is recommended for patients in whom the insomnia hinders the use of continuous positive airway pressure (CPAP) device. Another factor to be considered is that benzodiazepines

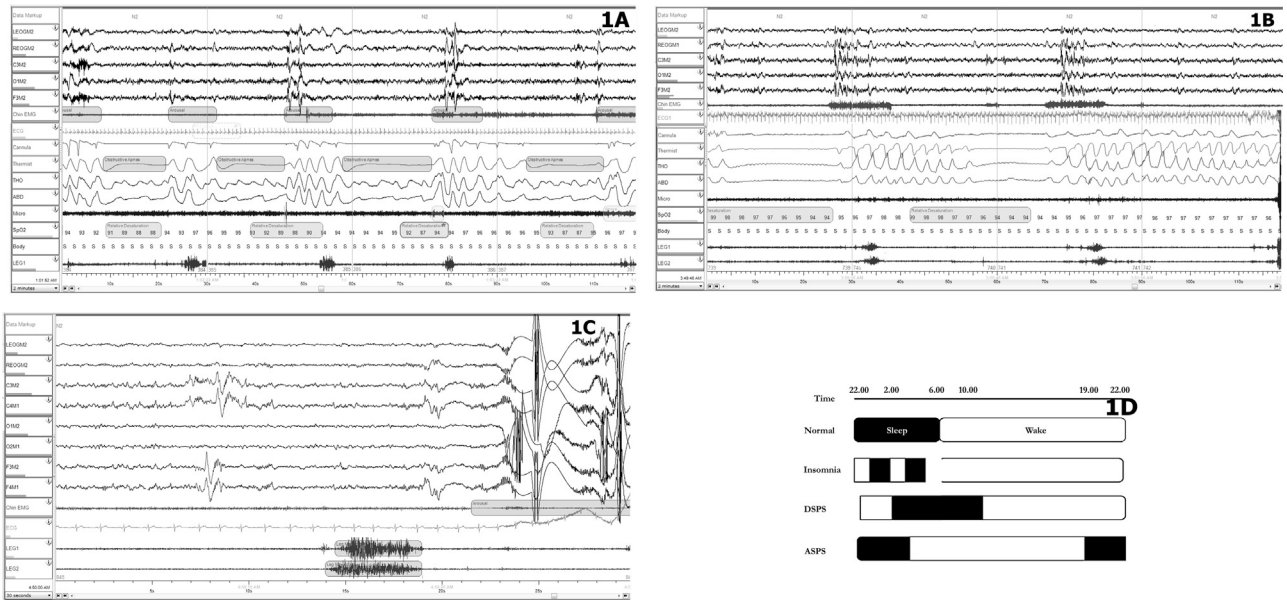

Fig. 1. Sleep disorders that may be mistaken for insomnia. (A) Two minute epoch showing four obstructive apneas (airflow, thorax and abdomen channels). Each apnea terminates with increment in EEG frequency suggesting arousal and limb movement (L Leg). (B) Two minute epoch showing two central sleep apneas (airflow, thorax and abdomen channels). Each apnea terminates with increment in EEG frequency suggesting arousal and limb movement (R Leg). (C) Thirty second epoch showing a limb movement during sleep (R Leg and L Leg). The movement was followed by appearance of alpha frequency in EEG channels. Presence of K complexes suggests stage 2 sleep. (D) Sleeping schedule of normal, insomnia, ASWPD and DSWPD subjects.

have a negative impact on respiratory mechanisms and therefore should be used cautiously for the treatment of insomnia comorbid with OSA (Berry et al., 1992; Maillard et al., 1992). Recently, non-benzodiazepines receptor agonists such as zolpidem, eszopiclone and zaleplon have become the main pharmacological therapeutic modality for insomnia. The influence of these "z" drugs on respiration is less pronounced than the effects of benzodiazepines (Sanger, 2004). Alternatively, CBT-I can be used in combination with CPAP treatment as a safe treatment option (Morgenthaler et al., 2006; Krakow et al., 2004).

5.3. Central sleep apnea (CSA)

Central sleep apnea is defined as cessation of breathing along with absence of respiratory efforts for at least 10 continuous seconds during sleep (Berry et al., 2014).Despite the fact that complaints of insomnia are not uncommon among patients with CSA, only a very limited number of studies have explored insomnia in this patient group. The first report describing the association between CSA and insomnia was published in 1976 (Guilleminault et al., 1976). Recently a case of CSA has been described in which the patient also had chronic sleep maintenance insomnia (Afaq et al., 2012). In another case, a patient with CSA also had chronic sleep maintenance insomnia and abnormal activity during sleep (Gupta et al., 2014). Patients with heart failure often have recurrent central sleep apnea, although it takes the form of Cheyene–Stokes breathing. Among patients with congestive heart failure, Sin et al. (1999) reported that the concurrent prevalence of central sleep apnea was 33%, 29% and 25%, using an AHI cutoff of 10, 15 and 20, respectively. Cheyne–Stokes respiration (CSR), which is a form of CSA, has been reported in 25–40% of patients with heart failure. Patients with CSR may complain of difficulties initiating and maintaining sleep (Javaheri, 2006; Oldenburg et al., 2007; Redeker and Stein, 2006). In a cross-sectional study that assessed sleep difficulties among patients with chronic heart failure, 24% of women and 23% of men reported having sleep maintenance insomnia compared to 10% and 8.5%, respectively, of normal subjects (Brostrom et al., 2004). In short, CSA must be ruled out in patients with chronic unexplained insomnia who are not responding to insomnia therapy. Unlike many OSA patients, patients with CSA are frequently thin; further, and additionally in contrast to OSA patients, they may not snore and typically have insomnia as the only presenting symptom (American Psychiatric Association, 2013; Quaranta et al., 1997).

5.4. Willis Ekbom's disease/Restless legs syndrome

Willis Ekbom's disease/Restless leg syndrome (WED/RLS) is characterized by an urge to move the legs, and is often associated with sensory symptoms e.g., pain in the legs, as well as sensations of tickling, tingling, stretching or bubbling (International Restless Legs Syndrome Study Group, 2012). The symptoms worsen during periods of rest e.g., at bedtime, but are relieved by counter stimulation in the form of massage, moving the legs or walking (International Restless Legs Syndrome Study Group, 2012). The symptoms follow a circadian course and are most severe during the

Table 1
Questionnaires available to screen common sleep disorders masquerading insomnia.

Disorder	Questionnaire name	Method	Approximate time (min)
OSA	STOP-Bang questionnaire (Chung et al., 2008)Berlin questionnaire (Netzer et al., 1999)	History and clinical examinationHistory and clinical examination	10–15
RLS	Cambridge–Hopkins questionnaire (CH-RLSq) (Allen et al., 2009)	History	15
Shift worker sleep disorder	Not applicable (Barger et al., 2012)	History	5

night. This symptom complex should be differentiated from that of several other conditions e.g., leg cramps, leg edema, habitual leg movements, positional discomfort, each of which is known to mimic WED/RLS (International Restless Legs Syndrome Study Group, 2012). While WED/RLS typically occurs at night, nearly half of affected patients have symptoms during the daytime as well, especially during periods of inactivity (Tzonova et al., 2012).

The symptoms of WED/RLS interfere with sleep and many WED/RLS patients complain of initial or middle insomnia (Anderson et al., 2013). Not only does WED/RLS lead to initial insomnia or multiple nocturnal awakenings, it is also impairs sleep quality by promoting sleep fragmentation (Hornyak et al., 2007) The periodic limb movements (PLMS) which are associated with WED/RLS produce micro-arousals which in turn worsen the quality of sleep (Fig. 1C) (Sforza et al., 1999). Affected patients therefore often complain of non-refreshing sleep upon awakening in the morning. The poor quality of sleep of these patients often leads to significant daytime symptoms e.g., fatigue, poor concentration or mood changes that may be mistaken for depression or somatoform disorder (Zhang et al., 2012). In such cases if antidepressants or sedating antipsychotics are prescribed for improving mood and sleep, the WED/RLS symptoms may actually worsen (ICSD-3). Screening tools for WED/RLS are also available (refer, Table 1).

5.5. Circadian rhythm sleep disorders

Circadian rhythm sleep disorders (CRSD) are a group of disorders in which the timing of the sleep and wake schedule is delayed or advanced relative to social norms (American Academy of Sleep Medicine, 2014). Despite the disruption to the sleep/wake schedule in affected patients, the total amount of time spent sleeping and sleep architecture may remain adequate (Fig. 1D). The exception to this generalization occurs in the case of shift workers who may never fully adjust to the abnormal scheduling imposed by their work shifts, and thus they may never experience an adequate amount of sleep (American Academy of Sleep Medicine, 2014). In either of these cases the final result is a misalignment between environmental and circadian timing, and thus a lack of correspondence between the abnormal sleep cycle and the desired sleep cycle, which lead to insomnia symptoms. The two most important circadian rhythm sleep disorders that are commonly seen in clinical practice are those in which the sleep phase is either advanced or delayed (American Academy of Sleep Medicine, 2014). Analyses of sleep diary reports, actigraphy or dim light melatonin onset test (DLMO) are reliable methods for ruling out these disorders (Wyatt et al., 2006) (Fig. 2).

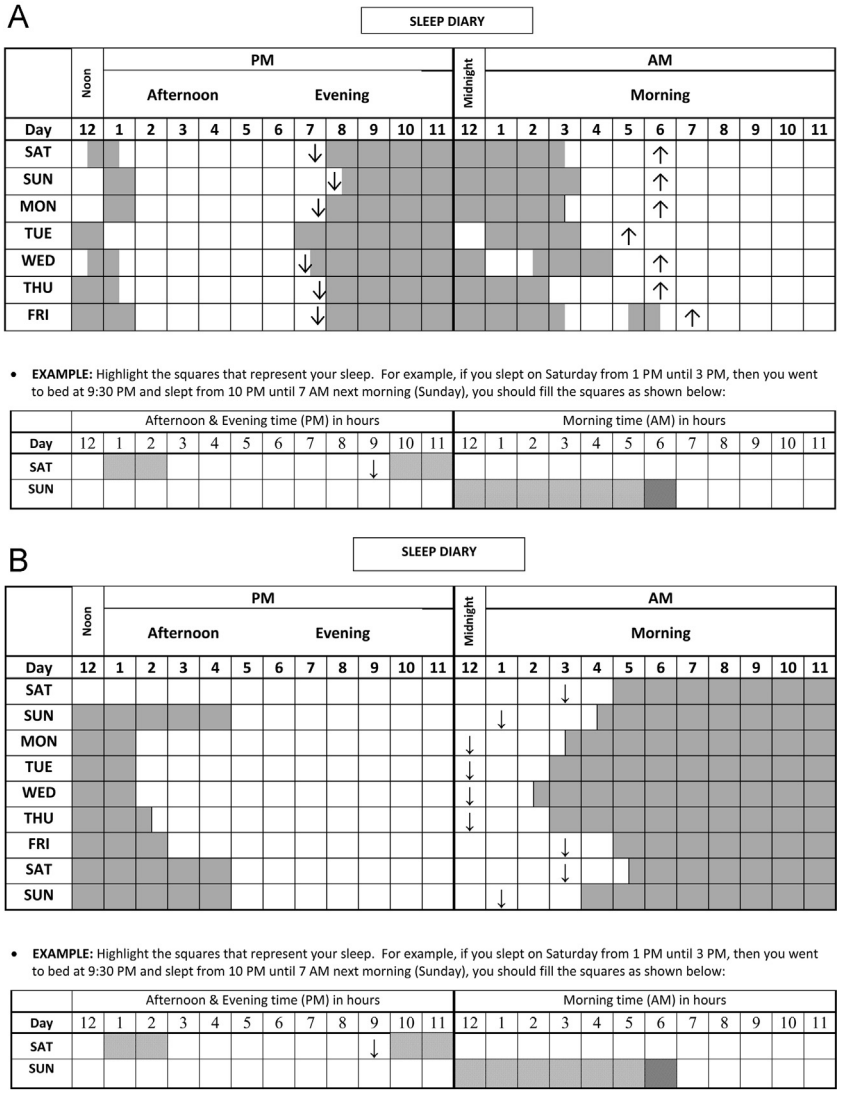

Fig. 2. Sleep diary showing common circadian rhythm sleep disorders. (A) Advance sleep–wake phase disorder. (B) Delayed sleep–wake phase disorder.

5.6. Advanced sleep–wake phase disorder

Advanced sleep–wake phase disorder is diagnosed when the sleep diary or actigraphy shows a consistent advanced sleep phase pattern for at least seven days (preferably 14 days) (American Academy of Sleep Medicine, 2014). In other words, patients with advanced sleep–wake phase disorder (ASWPD) go to bed early and wake up early and in between have adequate sleep unless they suffer from any other sleep disorder. Thus, affected patients experience a mismatch between the timing or phase of their biological clock and environmental timing. The principal symptom of these patients is a complaint of early morning or maintenance insomnia and excessive evening sleepiness. In other words, they often remain awake in the latter part of the night when other family members are asleep. Thus they spend time in bed staying awake and may be mistaken for terminal insomnia (Reid and Zee, 2009). However, unlike patients with insomnia, the sleep quality of ASWPD patients remains adequate i.e., it is similar to the premorbid duration and additionally they may not suffer from classical daytime symptoms of insomnia unless they try to follow the schedule that is desirable for their social activities. Advanced sleep phase has been found to occur much more frequently among the elderly thus leading to suggestions that the aging process itself may be a causal factor in the circadian disruption (Yoon et al., 2003a). This age group usually spends more time indoors, and is thus less physically active than younger adults (Yoon et al., 2003a). Thus, in ASWPD patients a combination of behavioral and environmental factors, including evening bright light therapy, less daytime exposure, the bright light and daytime naps may coalesce to promote the development of insomnia (Sack et al., 2007). These changes can be reliably picked by the actigraphy (Yoon et al., 2003b)

5.7. Delayed sleep–wake phase disorder (DSWPD)

Delayed sleep–wake phase disorder (DSWPD) is a condition caused by mismatch between the biological and environmental timing and is characterized by sleep-onset insomnia and delayed awakening in the morning (American Academy of Sleep Medicine, 2014). Individuals with DSWPD present with complaints of initial insomnia or morning hypersomnia (Reid and Zee, 2009). The 2014 ICSD-3 estimated that DSWPD is seen in approximately 10% of patients presenting in sleep clinics with recurrent insomnia complaints (American Academy of Sleep Medicine, 2014). However, similar to patients with advanced sleep phase syndrome, the total duration of time spent in sleep remains equivalent to premorbid levels. As a consequence, these patients do not present with symptoms of insomnia unless they try to modify their sleep according to social demands (American Academy of Sleep Medicine, 2014). This kind of sleep pattern is not uncommon during adolescence due to social and environmental reasons. While these symptoms are frequently encountered in adolescents and may mimic those of DSWPS, the resiliency of many affected individuals in re-adapting to a more normal sleep schedule, given adequate opportunities, suggests that the phenomenon is biologically, rather than socially driven. Current evidence regarding interventions supports the efficacy of timed melatonin administration to promote a corrective phase advance in patients with DSWPD. Timed bright light exposure treatment, based on the light phase response curve (PRC), may help in advancing sleep in patients with DSWPD. However, compliance with this treatment may be a significant problem (Sack et al., 2007). Use of sedatives to treat symptoms of insomnia may lead to substance abuse.

5.8. Parasomnias

Parasomnias are defined as unpleasant or undesirable behavioral or experiential phenomena that occur predominantly or exclusively during the sleep period e.g., sleep-walking or the experience of night terrors (American Academy of Sleep Medicine, 2014). These are known as disorders of arousal inasmuch as they occur during periods of micro-arousals from sleep (American Academy of Sleep Medicine, 2014). Parasomnias may themselves be an independent disorder or may sometimes be provoked or sustained by another sleep disorder such as OSA. Irrespective of the exact nature of the pathophysiology of parasomnias, their most relevant clinical feature is their association with micro-arousals and thus their tendency to worsen the quality of sleep (American Academy of Sleep Medicine, 2014). In the case of patients who suffer from recurrent parasomnias, there may also be complaints of non-refreshing sleep, one of the components of insomnia. For purposes of diagnostic screening it is therefore relevant to ascertain if patients who present with insomnia symptoms may also be experiencing parasomnias.

5.9. Sleep starts (hypnic jerks)

Sleep starts are characterized by a sudden jerky movement in the whole body which may occur along with the sensation of slipping or falling at the transition of wakefulness to sleep. Sometimes, a loud noise or a flash of light may be experienced subjectively. If it occurs repetitively, it may disrupt sleep quality and lead to sleep-onset insomnia (American Academy of Sleep Medicine, 2014). This phenomenon is not uncommon as it is often precipitated by stress, sleep deprivation or excessive caffeine or other stimulant intake. It affects all ages and both sexes.

5.10. Narcolepsy

While narcolepsy is commonly associated with hypersomnia, the condition is also characterized by multiple nocturnal awakenings and an increment of N1 sleep (Roth et al., 2013; Hong et al., 2000; Haimov and Lavie, 1997). The association of narcolepsy with difficulties in sleep maintenance has been known in this patient group for more than 20 years, and in many respects the condition shares commonalities with insomnia (Rosenthal et al., 1990). In addition to excessive daytime sleepiness, narcolepsy patients may present with sleep maintenance insomnia and difficulty consolidating sleep at night. This situation may be more relevant in young children in whom sleepiness may be difficult to assess.

It is known that narcoleptics often complain of excessive daytime sleepiness (EDS) and that they have a short sleep onset latency (SOL) and short REM sleep onset latency periods (SOREMPs) (Hong et al., 2000; Rosenthal et al., 1990; Li et al., 2007). These characteristics have been found more frequently in subjects with DQB1 0602* haplotype patients (Hong et al., 2000). Thus, narcolepsy may be viewed as a disorder of sleep/wake instability in which fragmented nocturnal sleep (non-refreshing sleep i.e., insomnia) and excessive daytime sleepiness are seen. These features imply a deficiency in orexin–hypocretin that is related to the pathogenesis of narcolepsy (Ferri et al., 2009).

6. Advantages of the changes

The changes brought in the DSM-5 will be helpful for the clinical practice. First, this system identifies that insomnia is not merely a symptom rather a comorbidity that occurs in context with psychiatric illness and medical disorders and requires independent attention (Reynolds and O'Hara, 2013). Furthermore,

insomnia may be an indicator of some medical disorders and they should be provided attention e.g., poor sleep quality may be an indirect evidence of neurodegenerative disorders, cardio-vascular illness or pulmonary disorders. Worsening of insomnia in a patient having insomnia for a long time should also arise suspicion towards underlying pathology not limited to above mentioned problems but also to the sleep hygiene, sleep environment, circadian rhythm and medications. This is another strength of the new system, that insomnia is seen across a spectrum and many scales are available to quantify the insomnia that can be used to assess severity and measure the progress across the therapy period (Reynolds and O'Hara, 2013). Lastly, this system not only helps in identifying the insomnia to its core, but also helps the mental health professionals to refer a patient to appropriate specialist e.g., neurologist, cardiologist, pulmonologist and sleep-physician so as to provide maximum benefit to the patients. Last, but not the least, it may be helpful in invoking the interest among mental health professionals to specialize in Sleep Medicine.

7. Conclusion

This article suggests that our understanding towards insomnia has changed. The nomenclature of insomnia, adopted in DSM-5 is based upon the recent evidences. These evidences suggest that insomnia occurring in context of psychiatric disorders and medical disorders is not just 'secondary', particularly when it assumes a chronic course. In all such conditions, insomnia should be given a separate diagnosis and should be treated as an independent disorder.

In addition, evidence has been reviewed showing that a number of sleep disorders may masquerade as insomnia. There are also situations in which chronic insomnia is diagnosed while underlying sleep disorders remain unnoticed. In cases where, based on screening or clinical interview, the presence of a comorbid sleep disorder is suspected, or when the insomnia symptoms persist despite adequate treatment, polysomnography is warranted (Cronlein et al., 2012). The diagnosis of insomnia in the context of other illnesses and sleep disorders is therefore complex and should be guided by an appreciation of its pathophysiology as well as by data obtained from polysomnography.

References

Afaq, T., Magalang, U.J., Das, A.M., 2012. An unusual cause of insomnia. Central sleep apnea (CSA). J. Clin. Sleep Med. 8 (5), 623–625.

Al-Jawder, S.E., BaHammam, A.S., 2012. Comorbid insomnia in sleep-related breathing disorders: an under-recognized association. Sleep Breath. 16 (2), 295–304.

Allen, R.P., Burchell, B.J., MacDonald, B., Hening, W.A., Earley, C.J., 2009. Validation of the self-completed Cambridge–Hopkins questionnaire (CH–RLSq) for ascertainment of restless legs syndrome (RLS) in a population survey. Sleep Med. 10 (10), 1097–1100.

Alotair, H., Bahammam, A., 2008. Gender differences in Saudi patients with obstructive sleep apnea. Sleep Breath. 12 (4), 323–329.

American Academy of Sleep Medicine, 2005. International Classification of Sleep Disorders: Diagnostic and Coding Manual, 2nd ed. American Academy of Sleep Medicine, Westchester, Illinois.

American Academy of Sleep Medicine, 2014. International Classification of Sleep Disorders: Diagnostic and Coding Manual, 3rd ed. Darian, IL.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Health Disorders, fifth ed. Washington.

Anderson, K., Jones, D.E., Wilton, K., Newton, J.L., 2013. Restless leg syndrome is a treatable cause of sleep disturbance and fatigue in primary biliary cirrhosis. Liver Int. 33 (2), 239–243.

Barger, L.K., Ogeil, R.P., Drake, C.L., O'Brien, C.S., Ng, K.T., Rajaratnam, S.M., 2012. Validation of a questionnaire to screen for shift work disorder. Sleep 35 (12), 1693–1703.

Bastien, C.H., St-Jean, G., Morin, C.M., Turcotte, I., Carrier, J., 2008. Chronic psychophysiological insomnia: hyperarousal and/or inhibition deficits? An ERPs investigation. Sleep 31 (6), 887–898.

Bastien, C.H., Turcotte, I., St-Jean, G., Morin, C.M., Carrier, J., 2013. Information processing varies between insomnia types: measures of N1 and P2 during the night. Behav. Sleep Med. 11 (1), 56–72.

Becker, P.M., Novak, M., 2014. Diagnosis, comorbidities, and management of restless legs syndrome. Curr. Med. Res. Opin. ([Epub ahead of print] PubMed PMID: 24805265).

Berry, R.B., Brooks, R., Gamaldo, C.E., Harding, S.M., Lioyd, R.M., Marcus, C.L., et al., 2014. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, 2.0.3.. Darien, Illinois.

Berry, R.B., McCasland, C.R., Light, R.W., 1992. The effect of triazolam on the arousal response to airway occlusion during sleep in normal subjects. Am. Rev. Respir. Dis. 146 (5), 1256–1260.

Bonnet, M.H., Arand, D.L., 2010. Hyperarousal and insomnia: state of the science. Sleep Med. Rev. 14 (1), 9–15.

Brostrom, A., Stromberg, A., Dahlstromm, U., Fridlund, B., 2004. Sleep difficulties, daytime sleepiness, and health-related quality of life in patients with chronic heart failure. J. Cardiovasc. Nurs. 19 (4), 234–242.

Buysse, D.J., Angst, J., Gamma, A., Ajdacic, V., Eich, D., Rössler, W., 2008. Prevalence, course, and comorbidity of insomnia and depression in young adults. Sleep 31 (4), 473–480.

Chung, F., Yegneswaran, B., Liao, P., Chung, S.A., Vairavanathan, S., Islam, S., Khajehdehi, A., Shapiro, C.M., 2008. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology 108 (5), 812–821.

Chung, K.F., 2005. Insomnia subtypes and their relationships to daytime sleepiness in patients with obstructive sleep apnea. Respiration 72 (5), 460–465.

Cronlein, T., Geisler, P., Langguth, B., Eichhammer, P., Jara, C., Pieh, C., Zulley, J., Hajak, G., 2012. Polysomnography reveals unexpectedly high rates of organic sleep disorders in patients with prediagnosed primary insomnia. Sleep Breath. 16 (4), 1097–1103.

Fernandez-Mendoza, J., Calhoun, S.L., Bixler, E.O., Karataraki, M., Liao, D., Vela-Bueno, A., Jose Ramos-Platon, M., Sauder, K.A., Basta, M., Vgontzas, A.N., 2011. Sleep misperception and chronic insomnia in the general population: role of objective sleep duration and psychological profiles. Psychosom. Med. 73 (1), 88–97.

Ferri, R., Franceschini, C., Zucconi, M., Drago, V., Manconi, M., Vandi, S., Bruni, O., Plazi, G., 2009. Sleep polygraphic study of children and adolescents with narcolepsy/cataplexy. Dev. Neuropsychol. 34 (5), 523–538.

Guilleminault, C., Eldridge, F.L., Phillips, J.R., Dement, W.C., 1976. Two occult causes of insomnia and their therapeutic problems. Arch. Gen. Psychiatry 33 (10), 1241–1245.

Guilleminault, C., Palombini, L., Poyares, D., Chowdhuri, S., 2002. Chronic insomnia, premenopausal women and sleep disordered breathing: part 2. Comparison of nondrug treatment trials in normal breathing and UARS post menopausal women complaining of chronic insomnia. J. Psychosom. Res. 53 (1), 617–623.

Gupta, R., Sindhwani, G., Rawat, J., Kesarwani, V., 2014. Idiopathic central sleep apnea: an Indian case with polysomnographic findings. Indian J. Chest Dis. Allied Sci. 56 (1), 41–44.

Gupta, R., Lahan, V., 2011. Insomnia associated with depressive disorder: primary, secondary, or mixed? Indian J. Psychol. Med. 33 (2), 123–128.

Haimov, I., Lavie, P., 1997. Circadian characteristics of sleep propensity function in healthy elderly: a comparison with young adults. Sleep 20 (4), 294–300.

Hong, S.C., Hayduk, R., Lim, J., Mignot, E., 2000. Clinical and polysomnographic features in DQB1*0602 positive and negative narcolepsy patients: results from the modafinil clinical trial. Sleep Med. 1 (1), 33–39.

Hornyak, M., Feige, B., Voderholzer, U., Philipsen, A., Riemann, D., 2007. Polysomnography findings in patients with restless legs syndrome and in healthy controls: a comparative observational study. Sleep 30 (7), 861–865.

International Restless Legs Syndrome Study Group, 2012. Revised IRLSSG diagnostic criteria. ⟨http://irlssgorg/diagnostic-criteria/2012.⟩ (accessed 10.09.13.).

Javaheri, S., 2006. Sleep disorders in systolic heart failure: a prospective study of 100 male patients. The final report. Int. J. Cardiol. 106 (1), 21–28.

Krakow, B., Melendrez, D., Lee, S.A., Warner, T.D., Clark, J.O., Sklar, D., 2004. Refractory insomnia and sleep-disordered breathing: a pilot study. Sleep Breath. 8 (1), 15–29.

Lavie, P., 2007. Insomnia and sleep-disordered breathing. Sleep Med. 8 (Suppl 4), S21–S25.

Li, J., Xu, Y., Dong, X.S., Han, X., He, Z.M., Lu, Y.H., Wang, F., He, Q.Y., Han, F., 2007. Changes of sleep architecture in patients with narcolepsy. Zhonghua Xue Za Zhi 87 (9), 619–621.

Lichstein, K.L., Riedel, B.W., Lester, K.W., Aguillard, R.N., 1999. Occult sleep apnea in a recruited sample of older adults with insomnia. J. Consult. Clin. Psychol. 67 (3), 405–410.

Maillard, D., Thiercelin, J.F., Fuseau, E., Rosenzweig, P., Attali, P., 1992. Effects of zolpidem versus diazepam and placebo on breathing control parameters in healthy human subjects. Int. J. Clin. Pharmacol. Res. 12 (1), 27–35.

Morgenthaler, T., Kramer, M., Alessi, C., Friedman, L., Boehlecke, B., Brown, T., Coleman, J., Kapur, V., Lee-Chiong, T., Owens, J., Pancer, J., Swick, T., American Academy of Sleep Medicine, 2006. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American academy of sleep medicine report. Sleep 29 (11), 1415–1419.

Netzer, N.C., Stoohs, R.A., Netzer, C.M., Clark, K., Strohl, K.P., 1999. Using the Berlin questionnaire to identify patients at risk for the sleep apnea syndrome. Ann. Intern. Med. 131 (7), 485–491.

Oldenburg, O., Lamp, B., Faber, L., Teschler, H., Horstkotte, D., Topfer, V., 2007. Sleep-disordered breathing in patients with symptomatic heart failure: a contemporary study of prevalence in and characteristics of 700 patients. Eur. J. Heart Fail. 9 (3), 251–257.

Pérusse, A.D., Turcotte, I., St-Jean, G., Ellis, J., Hudon, C., Bastien, C.H., 2013. Types of primary insomnia: is hyperarousal also present during napping? J. Clin. Sleep Med. 9 (12), 1273–1280.

Quaranta, A.J., D'Alonzo, G.E., Krachman, S.L., 1997. Cheyne-Stokes respiration during sleep in congestive heart failure. Chest 111 (2), 467–473.
Redeker, N.S., Stein, S., 2006. Characteristics of sleep in patients with stable heart failure versus a comparison group. Heart Lung 35 (4), 252–261.
Reid, K.J., Zee, P.C., 2009. Circadian rhythm disorders. Semin. Neurol. 29, 393–405.
Reynolds, C.F., O'Hara, R., 2013. DSM-5 sleep wake disorder classification: overview for use in clinical practice. Am. J. Psychiatry 170 (10), 1099–1101.
Rosenthal, L.D., Merlotti, L., Young, D.K., Zorick, F.J., Wittig, R.M., Roehrs, T.A., Roth, T., 1990. Subjective and polysomnographic characteristics of patients diagnosed with narcolepsy. Gen. Hosp. Psychiatry 12 (3), 191–197.
Roth, T., Dauvilliers, Y., Mignot, E., Montplaisir, J., Paul, J., Swick, T., Zee, P., 2013. Disrupted nighttime sleep in narcolepsy. J. Clin. Sleep Med. 9 (9), 955–965.
Roth, T., 2009. Comorbid insomnia: current directions and future challanges. Am. J. Manag. Care 15 (Suppl.), S6–S13.
Sack, R.L., Auckley, D., Auger, R.R., Carskadon, M.A., Wright Jr., K.P., Vitiello, M.V., Zhdanova, I.V., American Academy of Sleep Medicine, 2007. Circadian rhythm sleep disorders: part II, advanced sleep phase disorder, delayed sleep phase disorder, free-running disorder, and irregular sleep-wake rhythm. An American Academy of Sleep Medicine review. Sleep 30 (11), 1484–1501.
Sánchez-Ortuño, M.M., Edinger, J.D., 2012. Cognitive-behavioral therapy for the management of insomnia comorbid with mental disorders. Curr. Psychiatry Rep. 14 (5), 519–528.
Sanger, D.J., 2004. The pharmacology and mechanisms of action of new generation, non-benzodiazepine hypnotic agents. CNS Drugs 18 (Suppl 1), S9–S15.
Sforza, E., Nicolas, A., Lavigne, G., Gosselin, A., Petit, D., Montplaisir, J., 1999. EEG and cardiac activation during periodic leg movements in sleep: support for a hierarchy of arousal responses. Neurology 52 (4), 786–791.
Shimodera, S., Watanabe, N., Furukawa, T.A., Katsuki, F., Fujita, H., Sasaki, M., Perlis, M.L., 2011. Change in quality of life after brief behavioral therapy for insomnia in concurrent depression: analysis of the effects of a randomized controlled trial. J. Clin. Sleep Med. 10 (4), 433–439.
Sin, D.D., Fitzgerald, F., Parker, J.D., Newton, G., Floras, J.S., Bradley, T.D., 1999. Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. Am. J. Respir. Crit. Care Med. 160 (4), 1101–1106.
Taylor, D.J., Lichstein, K.L., Durrence, H.H., Riedel, B.W., Bush, A.J., 2005. Epidemiology of insomnia, depression, and anxiety. Sleep 28 (11), 1457–1464.
Turcotte, I., St-Jean, G., Bastien, C.H., 2011. Are individuals with paradoxical insomnia more hyperaroused than individuals with psychophysiological insomnia? Event-related potentials measures at the peri-onset of sleep. Int. J. Psychophysiol. 81 (3), 177–1790.
Tzonova, D., Larrosa, O., Calvo, E., Granizo, J.J., Williams, A.M., de la Llave, Y., García-Borreguero, D., 2012. Breakthrough symptoms during the daytime in patients with restless legs syndrome (Willis–Ekbom disease). Sleep Med. 13 (2), 151–155.
Unbehaun, T., Spiegelhalder, K., Hirscher, V., Riemann, D., 2010. Management of insomnia: update and new approaches. Nat. Sci. Sleep 2, 127–138.
van Mill, J.G., Hoogendijk, W.J., Vogelzangs, N., van Dyck, R., Penninx, B.W., 2010. Insomnia and sleep duration in a large cohort of patients with major depressive disorder and anxiety disorders. J. Clin. Psychiatry 71 (3), 239–246.
Wagley, J.N., Rybarczyk, B., Nay, W.T., Danish, S., Lund, H.G., 2013. Effectiveness of abbreviated CBT for insomnia in psychiatric outpatients: sleep and depression outcomes. J. Clin. Psychol. 69 (10), 1043–1055.
Watanabe, N., Furukawa, T.A., Shimodera, S., Morokuma, I., Katsuki, F., Fujita, H., Sasaki, M., Kawamura, C., Perlis, M.L., 2011. Brief behavioral therapy for refractory insomnia in residual depression: an assessor-blind, randomized controlled trial. J. Clin. Psychiatry 72 (12), 1651–1658.
Wickwire, E.M., Smith, M.T., Birnbaum, S., Collop, N.A., 2010. Sleep maintenance insomnia complaints predict poor CPAP adherence: a clinical case series. Sleep Med. 11 (8), 772–776.
Wyatt, J.K., Stepanski, E.J., Kirkby, J., 2006. Circadian phase in delayed sleep phase syndrome: predictors and temporal stability across multiple assessments. Sleep 29 (8), 1075–1080.
World Health Organization, 1992. The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva.
Yoon, I.Y., Kripke, D.F., Elliott, J.A., Youngstedt, S.D., Rex, K.M., Hauger, R.L., 2003a. Age-related changes of circadian rhythms and sleep-wake cycles. J. Am. Geriatr. Soc. 51 (8), 1085–1091.
Yoon, I.Y., Kripke, D.F., Youngstedt, S.D., Elliott, J.A., 2003b. Actigraphy suggests age-related differences in napping and nocturnal sleep. J. Sleep Res. 12 (2), 87–93.
Zhang, J., Lam, S.P., Li, S.X., Tang, N.L., Yu, M.W., Li, A.M., Wing, Y.K., 2012. Insomnia, sleep quality, pain, and somatic symptoms: sex differences and shared genetic components. Pain 153 (3), 666–673.